

Stig Willner

FACTORS CONTRIBUTING TO
STRUCTURAL SCOLIOSIS

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av

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by

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PREFACE

This dissertation is based on the following papers, references to which will be referred to in the text in Roman numerals.

- I Willner, S. (1972) Growth in height of children with scoliosis. (Accepted for publication in Acta Orthop. Scand)
- II Willner, S. (1972) A study of height, weight and menarche in girls with idiopathic structural scoliosis. (Accepted for publication in Acta Orthop. Scand).
- III Willner, S. (1972) The proportion of legs to trunk in girls with idiopathic structural scoliosis. (Accepted for publication in Acta Orthop. Scand).
- IV Willner, S. (1972) Pelvic obliquity and balance of the trunk in scoliosis. (Accepted for publication in Acta Orthop. Scand).

INTRODUCTION

The etiology and the pathogenesis of scoliosis have over the years attracted a great number of investigators. A review of the literature in this field was presented by Bouillet and Vincent (1967). James (1970 b) reviewing available data, stated that the pathogenesis is well known only in congenital scoliosis. In many pathologic conditions including neuro-muscular disturbance, scoliosis may be one symptom. These conditions may have a known etiology such as in poliomyelitis, in some cases, however, the basic etiology is unknown, for instance in syringomyelia or muscular dystrophy. However, in 80-90 % of the cases of structural scoliosis the etiology as well as the pathogenesis is unknown (Cobb 1948). This condition, the idiopathic structural scoliosis is found in children who are otherwise apparently healthy and normally developed. The research efforts invested in attempts to explain the etiology of scoliosis in this, the most important group will be reviewed briefly.

The primary skeletal hypothesis

A primary growth disturbance in the epiphyseal plates of the vertebral bodies has been suggested as the cause of structural idiopathic scoliosis (Haas 1939, Bisgard and Musselman 1940, Nachlas and Borden 1951, Arkin and Katz 1956, Ponseti and Shepard 1954 and others).

McCarroll and Costen (1960) found morphological differences between the growth cartilage cells in the epiphyseal plates between the convex and the concave aspects of the spine in cases with scoliosis and concluded that this was the primary cause of the deformity. Stilwell (1962) and Enneking and Harrington (1969), however, decided that these morphological changes were secondary.

Also asymmetric growth of the neuro-central epiphysis (the epiphyseal line in the junction of the vertebral body and the neural arch) has been suggested as a possible cause of structural scoliosis (Roaf 1960, Knutsson 1963 and 1966, Ottander 1963, James 1967, Karaharju 1967). Miniero (1969) noticed in animal experiments that the neuro-central epiphysis closed early on the concave side of the curve.

The nucleus pulposus has been suggested to be the site of the primary disturbance causing scoliosis. Perey (1963) demonstrated in three early cases of scoliosis asymmetry of the position of the nucleus and after removal of the nucleus pulposus the deformity decreased in these cases. Again Stilwell (1962) and McEven (1968) assumed that asymmetry of the nucleus pulposus merely was secondary to the structural changes in the spine. Roth (1969) suggested that the growth of the spine may be influenced by asymmetry in the anatomy of the nerve roots. Finally, Somerville (1952) and Roaf (1968) suggested that the scoliotic deformity may be caused by deviation from the normal relationship of growth between the neural arch and the vertebral body.

The neuro-muscular hypothesis

It has been suggested that idiopathic structural scoliosis may be the only remnant after a transient poliomyelitis (Colonna and Vom Saal 1941, Risser 1964). James (1967), however, pointed out that idiopathic structural scoliosis did not become less frequent after the introduction of polio vaccine. Gruca (1958) felt that idiopathic structural scoliosis was caused by muscular imbalance on the level of the primary curvature and this imbalance may be caused by a congenital, possibly inherited asymmetric segmental abnormality of the innervation of spinal muscles. Michele (1962) suggested that an asymmetrical action of the iliopsoas muscle was the primary cause of at least some cases of idiopathic scoliosis. Riddle and Roaf (1955) assumed that idiopathic scoliosis was primarily caused by an imbalance between left and right of the deep rotator and the transverse muscles.

Roaf (1968) suggested the following basic causes of muscular imbalance:

1. Primary myogenic disturbance (muscular dystrophy).
2. Lesions of the lower motor-neuron (poliomyelitis).
3. Lesions of the upper motor-neuron (cerebral palsy).
4. Disturbance of the postural mechanism (brain or mid-brain lesions).
5. Congenital spinal deformity.

Arkin (1950) suggested that the primary mechanism causing structural

scoliosis is soft-tissue tension rather than primary skeletal disorder and Olsen and Hamilton (1969) suggested that muscular imbalance is a necessary requirement for idiopathic scoliosis.

Garrett et al. (1961) found in cases which had been classified as idiopathic structural scoliosis a decreased force in paravertebral and intercostal muscles on the convex side of the spinal curve.

Lindahl and Raeder (1962) suggested that idiopathic scoliosis could be due to asymmetrical disturbance of growth of the intertransversal ligaments or muscles.

In animal experiments structural scoliosis of the type observed in man has been produced by resection of the posterior end of the ribs, the costo-transverse joints and/or destruction of the costo-transverse ligaments (Langenskiöld and Michelsson 1962, 1963, Michelsson 1965).

Wullstein (1902) and Carey (1932) found that in animal experiments structural scoliosis of the spine could be produced by maintaining the experimental animal in a position with a curved spine. Langenskiöld (1971) claimed that if a growing animal was forced into a curved position the growth of the muscles of the trunk on the concave side was disturbed leading to shortening and secondary skeletal deformity.

Denis Browne (1965) felt that in some instances infantile idiopathic scoliosis may have been caused by an abnormal intra-uterine posture. Gruca (1958) found morphological changes in spinal muscles by histological examination. James (1967 and 1970 b), however, failed to reveal any such changes and assumed that muscular imbalance has no part in the origin of idiopathic scoliosis.

Yamada et al (1969) tested the equilibrical function in children with structural scoliosis using proprioceptive reactions and ocular reflexes. The degree of equilibrical dysfunction was correlated to the severity of deformity and the signs of dysfunction disappeared as the growth of the spine was terminated.

Electromyographic findings in cases with structural scoliosis have given inconsistent results. Riddle and Roaf (1955) and Brussatis (1962) found that the electromyographic activity was greater in the convex than in the concave side of the spine, and claimed that the stronger muscles were usually on the convex side of the scoliosis. Zuk (1962), however, claimed, that the increased EMG activity on the convex side was more a sign of weakness of the muscles of this side. This increased activity was seen in idiopathic scoliosis under progress even with patients at rest. In cases without progress of the deformity this phenomenon was not found. Butter-

worth and James (1969) who had made the same observations as Zuk tried to use electromyography as a prognostic method of scoliosis.

Zuk (1965) observed in cases of idiopathic structural scoliosis electromyographic signs of peripheral paresis on the convex side in no less than 46 % of the cases. In an additional 42 % there were signs of increased excitability or contracture on the concave side and in the remaining cases no side difference in the electromyographic recordings were found.

Badger (1969) found electromyographic signs of myopathy on the convex side of the spine. Tokarowski (1965) found electromyographic changes on the concave side in idiopathic scoliosis similar to what is seen in muscular dystrophy and Saito (1964) found electromyographic signs of atrophic changes in the deep back muscles of the convex side.

Ooi (1966) detected in cases classified as idiopathic scoliosis electromyographic changes similar to what may be seen in paralytic cases.

So far electromyographic changes have not solved the problem as to whether the neuro-muscular condition is the primary cause of the deformity or not.

The metabolic hypothesis

Stearns et al. (1955) found a disturbance in catabolism of protein in children with idiopathic structural scoliosis. With increasing protein intake there was an increased excretion of amino acids, in particular taurin, but no abnormality in mineral metabolism was found. Schönenberger et al. (1962) noticed an increased excretion of taurin in scoliosis as well as in epiphyseolysis of the femoral head. Beals (1969) reviewed 79 cases of homocystinuria among which no less than 27 % had scoliosis. Also Frederich et al. (1963) noticed disturbances in the excretion of amino acids and suggested that changes in the protein metabolism were the primary cause of idiopathic structural scoliosis.

The serum content of varying fractions of protein components has also been analysed in cases with idiopathic structural scoliosis. Glauber et al. (1962) found an increased content of α_1 -globulin in a progressing idiopathic scoliosis. Böhmer and Nolte (1972) found a decreased amount of α_2 - and beta-globulin in children with structural idiopathic scoliosis in progress but not in children with congenital or paralytic scoliosis.

The importance of changes in the collagen metabolism has also been considered. Benson (1965) found in a limited series no convincing difference in the hydroxyproline excretion in children with and without scoliosis. Zorab et al. (1971), however, found an increased excretion of hydroxyproline in children with scoliosis. Franchi et al. (1963) found in children with idiopathic structural scoliosis an increase of anti-elastin inhibitors which was interpreted as a sign of degeneration of elastic structures.

Kyselka (1965) noticed in structural scoliosis in progression increased levels of phosphatase and of the phosphorus values and a decreased excretion of acid mucopolysaccharides in urine indicating an active osseous metabolic activity.

Lathyrism has been produced in the rat by adding beta-aminopropionitrile to the diet resulting in epiphyseal plate changes and scoliosis (Ponseti and Baird 1952 and Ponseti and Shepard 1954). Amato and Bombelli (1959) noted a disorganization of the cartilage columns in the epiphyseal plates of long bones in rats with lathyrism.

Yli-Pohja et al. (1958) noticed that the synthesis of hydroxyproline was impaired in lathyrism. Brodetti and Cauchoix (1962) found morphological evidence that the growth disorder of the epiphyseal plates in the spine of lathyric rabbits with scoliosis was primarily caused by vascular lesions and suggested a similar mechanism in human idiopathic scoliosis possibly as a genetic factor. They also found that the vertebrae Th VII-IX were less vascularized. Ponseti (1968) noticed an increase in total hexosamine concentration of iliac crest cartilage in patients with progressive adolescent idiopathic scoliosis.

Böhmer (1965) and Böhmer and Nolte (1972) established a significant decrease of the serum cholinesterase activity in children with idiopathic scoliosis in progress which became normal when the deformity was stationary.

The growth hormone level in girls with idiopathic scoliosis was measured by Misol et al. (1971) and found to fall within normal ranges.

Although not quite consistent the findings of metabolic deviations in children with idiopathic structural scoliosis suggest the presence of some in-born error of metabolism which effects the development of the skeleton, the neuro-muscular function or both.

Heredity

Faber (1936) presented observations indicating that idiopathic structural

scoliosis was a hereditary disease. Members of a family with a child suffering from scoliosis have according to Wynne-Davies (1968) a twenty times increased risk of scoliosis and this was true for infantile as well as adolescent scoliosis. Cowell et al. (1969) suggested that idiopathic scoliosis was inherited as a sex linked dominant condition of varying penetration. James (1970 a) assumed that adolescent idiopathic scoliosis in girls is more of a dominant than a recessive inheritance.

In twin studies Esteve (1958) found that scoliosis in identical twins, was not necessarily of equal severity but identical in shape and level. Murdoch (1959) and Berquet (1966) also reported scoliosis in identical twins. On the other hand Fisher and DeGeorge (1967) suggested that environmental factors predominate over genetic.

Recently, the Scoliosis Research Society (1972) discussed the substitution of "idiopathic" scoliosis with "genetic" scoliosis.

Growth

The process of maturation in man is influenced by hereditary and environmental factors. Maturity may be defined as the result of growth and differentiation (Goldberg 1955). Height, weight and menarche are the most commonly used measures of maturity. None of these variables, however, provide a sufficient estimate of the process of maturation and variables such as skeletal age and physiological age have therefore been introduced. In man growth is not a linear function. The growth rate is most rapid in the newborn, the rate decreases during the first four years of life and thereafter remains constant to the time of the prepuberal growth spurt. In some children there is a temporarily increased rate of growth during the sixth to eighth years of life, the so-called juveniline or mid-growth spurt (Grubb 1942). The existence of the mid-growth spurt has been debated by Tanner (1962). Frisch and Revelle (1971 a) found that 6.5 % of all girls and 8.4 % of all boys had a constant rate of growth throughout their childhood without the usual rapid increase in rate about the age of 10.

The prepuberal growth spurt in girls usually starts at the age of 10 and in boys about two years later. Frisch and Revelle (1971 a) found that the age at the onset of the growth spurt for girls was 9.6 ± 1.0 , and for boys 11.7 ± 0.9 and that the first sign of changes in the secondary sexual characters came about one year later. The duration of adolescence is on the average three years from onset to completion. Most girls have reached the maximal growth rate before breast and pubic hair are fully developed and all girls reach menarche after the point of maximum growth rate

(Marshall and Tanner 1969). The same authors determined the maximum growth rate in girls to 12.1 ± 0.9 and menarche to 13.5 ± 1.0 . The maximum growth rate in boys was determined to occur at the age of 14 (Tanner 1962). Weight and height follow similar patterns but although there is a rapidly increasing weight during the first year of life, the most rapid increase is seen during adolescence. From the age of two to the onset of puberty the increase of weight is fairly constant but somewhat accelerated at the age of 6-8 (Frisch and Revelle 1971 a). The onset of the prepuberal weight spurt coincides largely with that of height, 9.5 ± 1.0 for girls and 11.6 ± 0.9 for boys (Frisch and Revelle 1971 a). Also here the maximum rate of increase comes before menarche (Simmons and Greulich 1943 and Frisch and Revelle 1969). The latter authors (1969, 1970, 1971 b) also found that girls with early and late menarche differ in height but not in weight. The average weight at menarche was 48 kg and the average menarcheal age 12.9 years. The average height was 158.5 and in girls with early and late menarche 158 and 162 cm respectively. Tanner (1962) noted that girls with an early menarche were heavier per unit height than girls with a late menarche.

When comparing the sexes only small differences have been found between girls and boys before onset of adolescence. Cates and Goodwin (1936) claimed that boys were slightly taller during the first year of life whereas Shuttleworth (1938) and Hammond (1957) found no differences between the ages one and nine, but between 9 and 12 girls were heavier and taller (Tanner 1962, Marshall and Tanner 1968). The menarcheal age has shown a secular decreasing trend (Broman et al. 1942, Tanner 1966, Zacharias and Wurtman 1969 and others). Tanner (1962) demonstrated that the age at menarche decreased between 1830 and 1960 by about four months per decade in Western Europe. Among Scandinavian data on menarcheal age Samuelsson (1943), Sweden, 15.0 years, Lenner (1944), Sweden, 14.5 years, and Andersen (1968), Denmark, 13.3 years may be mentioned. The secular trend seems to apply also to other aspects of maturation. Tanner (1962) and many others found that the height has increased in the last century and that adolescence comes earlier. The increase in height in children is even greater than in adults. The prepuberal growth spurt is more intensive if it occurs earlier in life (Boas 1932, Shuttleworth 1939, Simmons and Greulich 1943, Tanner 1962). In these individuals the puberal process also passes more quickly. Children who are taller before puberty begin their growth spurt earlier than those who are initially shorter (Boas 1932). In adult women, however, there is no difference in height between those who have early and late menarche (Tanner 1962). On the contrary, girls with a late menarche have been reported to be taller (Shuttleworth 1939).

The relationship between growth and development of scoliosis was pointed

out by Bampfield (1824) and is now well established. Adams (1865) stated that rapid growth increased the risk of progress of scoliosis and that progress was rarely seen after the cessation of growth. Ridlon (1890) found that in un-treated scoliosis the deformity increased throughout the period of growth and Port (1903 and 1925) pointed out that structural scoliosis occurred only in childhood and that the rate of growth in height and the progress of scoliosis were related regardless of the etiology of the deformity. Schede (1938) demonstrated that the risk of progress was greatest during the period of rapid growth - age 13 and 14 - and Farkas (1954) claimed that idiopathic scoliosis occurred at a very early age but remained insignificant to prepuberty at the age of 10-12 and from there on progressed rapidly. Calvo (1957) found that progress of scoliosis occurred if the rate of growth of the spine - measured from the 8th to the 12th thoracic vertebra - exceeded 0.3 mm per month and that progress usually stopped at a skeletal age of 15 and the growth of the spine at a skeletal age of 16.

Duthie (1959) demonstrated that in infantile scoliosis there was a positive relationship between the velocity of growth in early postnatal life and the progress of the deformity. Ponseti and Friedman (1950) and James (1951) suggested that basically two factors influence the progress namely: the age at onset and the level of the curve and that scoliosis was most frequently diagnosed in the age groups 0-2, 5-7 and after 10, indicating a relationship between onset of scoliosis and rapid growth. Between 8 and 9 when the growth was less rapid scoliosis was rarely diagnosed. Scheier (1967) presented similar observations. Duval-Beaupère et al. (1967 a, 1967 b, 1970 and 1971) made prospective studies of children with scoliosis and found that the deformity increased linearly in two phases, a slow phase up to the age of 10 and thereafter a rapid phase. The same was true for paralytic and idiopathic cases. The second rapid phase was linear also during the prepuberal growth spurt and continued three years after menarche. They suggest that there is no relationship between progress and rate of growth. There was no difference between the rate of growth in children with and without scoliosis, nor was there any difference in height. Therefore tall children should not be more predisposed for scoliosis or develop a more severe scoliosis than children destined to be shorter.

MATERIAL AND METHODS

Definitions

The terminology used in this presentation follows closely that of the Terminology Committee of the Scoliosis Research Society (1969).

Scoliosis was defined as a lateral deviation of the spine between the C VII and L V levels with one or more curves along this part of the spine.

Functional scoliosis was defined as a lateral deviation found in the spine with the child standing which could be actively and passively reduced and which was not associated with rotational deformity.

Static or compensatory scoliosis includes cases in which the deformity compensates for a pelvic obliquity. Cases without obliquity are referred to as postural scoliosis. Functional scoliosis was corrected in sitting or lying position.

Structural scoliosis was defined as a deformity which could not be actively or passively reduced. Signs of rotational deformity were required. The deformity was not corrected in sitting or lying position.

Early onset - diagnosis before the age of 10.

Late onset - diagnosis from the age of 10. The age limits were chosen because of the prepuberal growth spurt in girls which usually starts at this age. The group with late onset is identical to adolescent scoliosis and early onset includes infantile and juvenile cases.

C-shaped scoliosis - one single curve of the spine.

S-shaped scoliosis - two or more curves of the spine.

Left and right convex - in C-shaped cases defined as the side of the convexity and in S-shaped cases as the side of the convexity of the proximal curve.

Pelvic obliquity was defined as a tilting of the pelvis (left-sided obliquity = tilting to the left) visually estimated to exceed one cm judged from the position of the iliac crests.

Balance of the trunk was defined as a deviation of the centre of the pelvis from the plumb line through the seventh cervical spinal process.

Age refers to chronological age.

The Linköping material

During the years 1957-1967 the school physicians in the city of Linköping and its immediate surroundings were encouraged to refer all children with a suspicion of scoliosis, even including slight deformities. If the diagnosis was confirmed when the child was seen in the Orthopaedic Department the case was included for further study. When the material was surveyed data pertinent to growth studies were sufficient in 1,616 children, 905 girls and 711 boys. For the study of pelvic obliquity and its relationship to the shape of the scoliosis sufficient data were available in 1,979 children, 1,089 girls and 890 boys. In both sets of children about 20 % had S-shaped scoliosis.

The Gothenburg material

Between the years 1963-1971, 311 girls with structural idiopathic scoliosis were referred to the Department of Orthopaedic Surgery in the city of Gothenburg. The cases were drawn from southern and western Sweden. One hundred and fourteen girls with a less severe deformity ($< 20^{\circ}$ according to Cobb 1948) provided data on height only. In the remaining 197 cases which had been admitted to the Orthopaedic Department prospective measurements of height and weight were available.

Control children

For comparison of heights control children were selected from the schools in Linköping. They were born 1953-1954 and longitudinal height measurements had been carried out between the ages of 7 and 15. There were 125 boys and 116 girls. The schools were chosen in order to be representative

of the socio-economic situation in the city. For comparison to older age groups high-school girls aged 18-19 and military service men, aged 19 were included, altogether 250 individuals. Except for the original sample of school children two additional samples were collected from schools in Linköping in which the measurements had not been performed annually but with at least four height measurements per individual, viz. at 7, 10, 11 and 13. These two samples did not differ in height from the original sample. Also, the control children were compared to three other contemporary studies of growth in children, all from Scandinavia. There was good agreement between the controls in the present study and data obtained elsewhere. As a consequence the control children obtained in Linköping were also used in the study of the Gothenburg material.

In the control children sitting heights had not been measured. Therefore, an additional sample of girls between 11 and 16 was measured in 1972 in the schools of the city of Malmö. Later on also data by Karlberg 1972 became available and were used for comparison with the Malmö control material.

For comparison of weights control data obtained by Karlberg 1972 were used.

Methods

In the Linköping material the data were obtained entirely by clinical examination. The child was examined in standing position, the spinal processes were located and marked on the skin. If the spinal processes deviated one cm or more from a straight line combining the spinal process of the C VII and the crena ani the case was accepted as scoliosis (figure 1). Although the severity of scoliosis expressed as the maximum deviation from the straight line was demonstrated to be related to the angulation the former variable was not further used (figure 2). The balance of the trunk was estimated from the position of a plumb-line suspended from the spinal process of the C VII at the level of the pelvis, to the right, to the left or in the mid-line. Finally the position of the pelvis was determined by identifying the iliac crests and left or right tilt was recorded with the child standing with extended knees and equal load on both legs. The presence or absence of rotational deformity was determined with the child in a forward-bending position. By changing the position of the child it was decided whether the deformity could be reduced or not.

Finally, the total height in standing and as previously mentioned in some



Fig. 1. The use of a plumb line to establish lateral deviation of the spine and balance of the trunk.

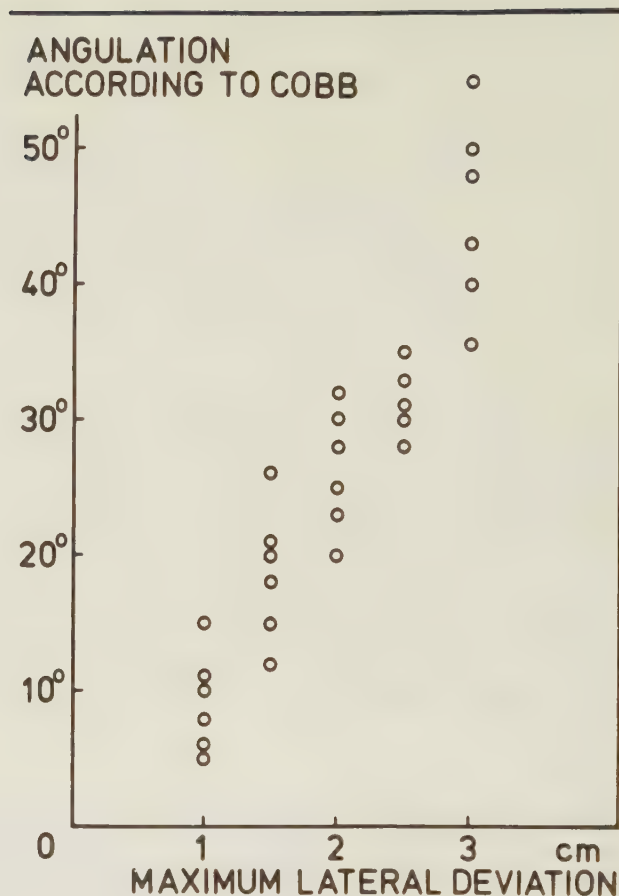


Fig. 2. The relationship between lateral deviation according to figure 1 and the curvature of the spine measured according to Cobb (1948). The cases were selected to represent increasing degrees of deformity but otherwise at random.

of the children also the sitting height (head plus trunk) was measured. This procedure was repeated every time the child was examined.

It was not feasible to examine roentgenographically all the children involved in the Linköping study. In 448 children, however, roentgenograms were obtained either in order to be able to follow a scoliosis which was

suspected to progress or to confirm a doubtful diagnosis. In all these cases a clinical diagnosis of scoliosis was confirmed and the localization in S-shaped cases agreed with the clinical findings. In 150 consecutive cases of clinically C-shaped scoliosis roentgenograms were taken in order to make a systematic study of the method of clinical classification. The diagnosis of scoliosis was confirmed in all instances but in seven children an S-shaped scoliosis was demonstrated which clinically had appeared to be C-shaped.

In the Gothenburg material radiograms and frequently repeated roentgen-examinations were available. The severity of scoliosis was in this study classified according to Cobb (1948) and the height measurements were recorded both as the uncorrected height and corrected for the degree of scoliosis according to the method of Bjure et al. (1968). In the Linköping, series radiograms were in most instances not available and the clinical classification of severity was not considered sufficiently accurate to allow for height corrections. The girls of the Gothenburg series were repeatedly interviewed with regard to the first menstruation in order to obtain an exact menarcheal age.

The data obtained in the height studies are cross-sectional and longitudinal and as the two methods do not necessarily yield the same information the calculations have been carried out as follows:

For studies of growth rate longitudinal data only were used. This presented no problem in the controls who had been measured between the ages of 7 and 16. In scoliotic cases, however, only those with at least two measurements could be included in this study, those data are still being longitudinal (mixed longitudinal study) although more individuals were needed to cover the age span with a sufficient number of observations.

In the comparisons of heights the longitudinal data of the controls were compared to cross-sectional data of children with scoliosis. However, the statistical methods applied will not be invalidated by the fact that the observations of the control children are only partially independent.

RESULTS

Height

Children with scoliosis were taller than children without scoliosis, boys as well as girls.

This was found in cases with functional as well as structural scoliosis (tables VII and VIII) (I).

When the cases of idiopathic structural scoliosis in girls were subdivided in groups of severity according to the angulation of the primary curve ($< 20^\circ$, 20° - 40° and $> 40^\circ$) there was no significant difference in corrected height between the sets (table IV, figure 2) (II).

There was evidence that the observed increase in height in idiopathic structural scoliosis exists only in the adolescent group and that children with an onset of their scoliosis before the age of ten (infantile and juvenile scoliosis) do not deviate from normal height. This difference could be studied in girls only (table V, figure 3) (II).

In the larger series (Linköping) where radiograms were usually not available there was no correction for the possible shortening of the trunk caused by the deformity. The girls with idiopathic structural scoliosis in the Gothenburg material deviated from normal height even before correction for the curvature of the spine and not even the most severe cases could be demonstrated to be shorter than the control cases. With correction for the curvature the difference in height between controls and scoliotic children was considerable (II).

The un-corrected height, however, tended to approach that of the control children and in the Linköping material the oldest girls, over the age of 16 could not be demonstrated to deviate significantly from the controls.

The pre-puberal growth spurt could not be identified in girls with functional and structural scoliosis, but the pattern of growth in the years before puberty differed significantly between girls with scoliosis and controls. In boys this deviation was not significant (tables V and VI, figure 5 and 6) (I)

The sitting height was also increased above normal in girls with idiopathic structural adolescent scoliosis (table I and figure 3) (III). The sitting height compared to the total height demonstrated that the proportions of the body, i. e. the relationship of spine to legs, was undisturbed in the girls (table II) (III).

Weight

Girls with idiopathic structural scoliosis, at least after the age of 13, were leaner than controls (table VII, figure 5) (II).

Menarche

The average menarcheal age in girls with idiopathic structural scoliosis was 13.1 ± 1.2 ¹⁾ years which does not deviate from the expected. However, within the group of girls with structural idiopathic scoliosis (the Gothenburg material) there was a tendency towards a positive relationship between early menarche and early diagnosis of scoliosis. A possible interpretation is that the deviation of the growth pattern in girls with scoliosis usually is unrelated to menarche or menarcheal age but that in some instances or in a subset of the group there is such a tendency (figure 7) (II).

Pelvic obliquity and shape of the scoliosis

Pelvic obliquity, that is a tilting of 1 cm or more to one side or the other, was found in 87 % of the cases in the Linköping series. Among these cases right-sided tilting was recorded in less than one fourth of the young children. With increasing age, however, the left-sided dominance decreased (tables III and V) (IV).

There was an agreement between the tilting of the pelvis and the shape of the spine so that in C-shaped cases the convexity usually was to the left if there was a left-sided pelvic obliquity and vice versa. This agreement was better in cases with left-sided tilting than in cases with right-sided tilting. The agreement was better in C-shaped functional cases than in S-shaped, structural cases but also in the latter group there was agreement between the tilting of the pelvis and the convexity of the lower curve (table IV) (IV).

1) Average $\pm$ standard deviation.

Burwell (1971) suggested a distinction between factors causing structural scoliosis namely: a) primary causes, b) secondary aggravation factors and c) limiting factors.

So far the primary cause, the basic etiology of idiopathic structural scoliosis is unknown and the findings of the present study will be discussed as possible, aggravating factors. The relationship between growth and scoliosis has been well documented as demonstrated above and the more recent opinions on this subject have been presented by Zorab (1971). There is no proof in the literature that children with scoliosis are taller or shorter than normal children. Duval-Beaupère (1971) stated that children with scoliosis were normal in this respect. The findings of the present study, obtained from two independent sources and related to controls, the representability of which has been tested, show that children with scoliosis, functional or structural, are taller than the average Swedish population. It is not possible to establish whether these children are constitutionally taller from early childhood or have been subjected to an early growth spurt at the time of the onset of the deformity. The hypothesis may be formulated that above average height in a child or deviation of growth pattern at the time of the onset of the deformity is an aggravating secondary factor which contributes to the production of the disease. Children with a C-shaped functional scoliosis are also taller than normal but do usually not develop the structural changes, on the opposite there is a tendency to restauration to normal. These children, having only one of the aggravating factors and lacking the unknown basic requirement do not develop the disease.

As previously pointed out (Tanner 1962) and others the early growth spurt usually results in a more intense growth. Jackson (1969) claimed that rapid growth increases the risk of asymmetric development of the body. Under the assumption that an early and rapid growth spurt in children with scoliosis is the cause of their above average height this deviation from normal per se might contribute to the development of scoliosis.

The sexual maturity as reflected in menarcheal age does not seem to deviate from the normal in most instances of structural idiopathic scoliosis. Within the group, however, there is a tendency towards a relationship between the age at onset or diagnosis and the age at menarche possibly indicating the presence of subsets where the coupling of growth spurt and menarcheal age is of some importance.

With regard to weight girls with idiopathic structural scoliosis do not deviate from normal before the age of 13. After that age girls with scoliosis are leaner. This finding defies at the present time interpretation and should be followed up by additional anthropometrical studies.

In children with the deformity diagnosed before the age of 10 there was no deviation in height from that of the control group. This group includes infantile and juvenile idiopathic structural scoliosis which could not at the time of diagnosis be separated. It has previously been pointed out (James 1954 and 1967) that infantile scoliosis in several aspects differs from the adolescent group. It is more frequent in boys. The convexity is more frequently to the left than to the right and the prognosis is more favourable. It must be suspected that infantile scoliosis is an entity different from the adolescent type and this is in the present study confirmed by the finding of the difference in height between the two.

It is an old observation that in scoliosis, also structural scoliosis, there is an agreement between pelvic tilting and the side of the curvature of the spine (Haglund 1923, Romich 1927 and Ingelmark 1943) and the findings of these investigators are confirmed in the present study.

Pelvic tilting may be considered as a sign of leg length difference. Other explanations to obliquity of the pelvis are not entirely ruled out, but in 150 consecutive radiograms of children in the Linköping group there was not one single case of asymmetric development of the pelvis. Haglund (1923), Romich (1927), Ingelmark (1943), Hult (1954) and Groh (1966) have demonstrated that during childhood in cases of unequal leg length the left leg is shorter but that in adults the right leg is the shortest. This agrees well with the findings in the present study in that there is a majority of left-sided pelvic tilting which becomes less predominant during the latter part of the growth period. As the leg length difference in most of the children in the Linköping material was the cause of the deformity (static scoliosis) and thus the basis for the primary selection it must also be suspected that children with scoliosis more often than other children have a detectable leg length difference. This implies that height above the average is associated with leg length discrepancy.

Although it cannot be denied that there is a relationship between pelvic tilting and the shape of an adolescent idiopathic structural scoliosis, pelvic tilting has been rejected as a cause of or even as a contributing factor to the deformity (Donaldson 1963, James 1967).

Height above the average is not sufficient to produce functional or structural scoliosis. In cases with structural scoliosis height cannot be de-

monstrated to influence the degree of the deformity. Therefore the deviation in height observed in children with scoliosis is probably a secondary requirement needed in some instances to trigger the mechanism of scoliosis development rather than an aggravating factor. As leg length difference and pelvic obliquity on one side and height on the other side appear to be related, the importance of a tilting pelvis as a secondary triggering factor cannot be entirely ruled out.

SUMMARY AND CONCLUSIONS

In about 1,900 children with functional or structural scoliosis of varying degree the position of the pelvis and the shape of the scoliosis were recorded. In about 1,600 of the children the heights were also recorded and in most instances followed prospectively. In an additional sample of about 300 girls with structural idiopathic scoliosis the height, weight and in part of the sample the menarcheal age were recorded. The data with regard to height were compared to a prospective study of the height of a control material of school-children.

1. Children with scoliosis are taller than children without scoliosis, boys as well as girls and cases with functional as well as cases with structural scoliosis.
2. The height did not vary with the degree of structural scoliosis.
3. Children with an early onset and diagnosis of structural scoliosis - before the age of 10 - were classified as infantile or juvenile cases and did not deviate in height from the control children.
4. The prepuberal growth spurt could not be identified in girls with functional or structural scoliosis and a similar tendency was - although not as obvious - found in boys. It is suggested that the period of rapid growth has occurred prior to the diagnosis of the condition indicating a deviation in the growth pattern of these children.
5. The relationship between the leg length and the height of trunk plus head remained the same in the girls with structural scoliosis.
6. Girls with structural scoliosis were at least after puberty leaner than normal girls.
7. No deviation in average menarcheal age could be demonstrated although within the group there was a slightly positive relationship between the age at diagnosis of the deformity and menarcheal age.
8. In accordance with previous investigations it was found that a left-sided tilting of the pelvis in children with functional or structural scoliosis

was associated with a left-convex curve in cases with C-shaped deformity and a left-convex lower curve in children with an S-shaped deformity.

9. The agreement between curve and pelvic tilting was better in left-sided than in right-sided cases.

10. The agreement was better in children with C-shaped, functional scoliosis than in children with S-shaped, structural scoliosis.

11. In older age groups the left-sided pelvic tilting was still dominant but less so than in young children.

It is suggested that a deviating growth pattern resulting in taller stature is a secondary factor which in the presence of an unknown basic requirement may trigger the development of structural scoliosis. Obliquity of the pelvis appears to be related to this deviation in growth and, also, to the shape of structural scoliosis.

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